Social cohesion, depression, and substance use severity among young men: Cross-sectional and longitudinal analyses from a Swiss cohort

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Abstract: INTRODUCTION Social cohesion, depression, and problematic substance use are intertwined and poorly understood.This study aimed to examine cross-sectional and longitudinal associations between social cohesion, depression and problematic substance use amongyoung men, age 21-25. METHODS We used 2nd wave (t_1, 2012-2014, N = 6020) and 3rd wave (t_2, 2016-2018) data from the on-going Swiss CohortStudy on Substance Use Risk Factors (C-SURF), assessing social cohesion, depression, and severity of alcohol, nicotine and cannabis use during both waves. Structural Equation Models (SEMs) were employed to examine pathways in both waves under the framework of longitudinal analysis. RESULTS Social cohesion was directly associated with depression and problem nicotine and cannabis use and indirectly associated with problem alcohol, nicotine and cannabis use through depression at both t1 and t2. Social cohesion exerted direct effects on nicotine use and cannabis use severity, but not on alcohol use severity. Social cohesion had indirect effects on problem use of all three substances, mediated via depression. The predictive direction was from depression to substance use, rather than vice versa. CONCLUSIONS Higher social cohesion at an early age appears to protect young males from depression and problematic substance use later in life. However, once problematic substance use is established, the direct effect of social cohesion diminishes and is mediated through personal depression. Therefore, promoting a more cohesiveneighborhood in childhood or adolescenthood could play an important role preventing depression and more severe substance use behaviors.

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Social cohesion, depression, and substance use severity among young men: Cross-sectional and longitudinal analyses from a Swiss cohort

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HIGHLIGHTS

- Social cohesion has direct effects on nicotine use and cannabis use severity, but not on alcohol use severity.
- Social cohesion has indirect effects on problem use of all three substances via depression.
- The predictive direction is from depression to substance use, rather than vice versa.

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ABSTRACT

Introduction: Social cohesion, depression, and problematic substance use are intertwined and poorly understood. This study aimed to examine cross-sectional and longitudinal associations between social cohesion, depression and problematic substance use among young men, age 21–25.

Methods: We used 2nd wave (t_1, 2012–2014, N = 6020) and 3rd wave (t_2, 2016–2018) data from the on-going Swiss Cohort Study on Substance Use Risk Factors (C-SURF), assessing social cohesion, depression, and severity of alcohol, nicotine and cannabis use during both waves. Structural Equation Models (SEMs) were employed to examine pathways in both waves under the framework of longitudinal analysis.

Results: Social cohesion was directly associated with depression and problem nicotine and cannabis use and indirectly associated with problem alcohol, nicotine and cannabis use through depression at both t1 and t2. Social cohesion exerted direct effects on nicotine use and cannabis use severity, but not on alcohol use severity. Social cohesion had indirect effects on problem use of all three substances, mediated via depression. The predictive direction was from depression to substance use, rather than vice versa.

Conclusions: Higher social cohesion at an early age appears to protect young males from depression and problematic substance use later in life. However, once problematic substance use is established, the direct effect of social cohesion diminishes and is mediated through personal depression. Therefore, promoting a more cohesive neighborhood in childhood or adolescenthood could play an important role preventing depression and more severe substance use behaviors.

1. Introduction

Substance use among adolescents and young adults remains one of Europe's most entrenched and costly health problems, and is the leading cause of mortality among young age groups worldwide, accounting for an estimated 35.3% of all deaths in 15 to 29-year-old men in developed countries (Toumbourou et al., 2007). Alcohol, tobacco, and cannabis are the three most frequently used substances. Several studies have focused on how to reduce the prevalence of risky substance use and corresponding substance use disorder (SUD). Social cohesion is often discussed, due to its effects through the community or neighborhood (Alcalá, Sharif, & Albert, 2016).

Social cohesion refers to the extent of connectedness and solidarity among groups in a society, and may be described as (1) the absence of

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latent social conflict; and (2) the presence of strong social bonds (Kawachi & Berkman, 2000). It includes trust of, attachment to, and tolerance of or respect for others (Stafford et al., 2003). There is increasing evidence on the protective effects of social cohesion against substance use, and especially against tobacco and cannabis. Higher social cohesion was found to reduce the likelihoods of both tobacco and cannabis use, as well as the amounts consumed (Lin, Witten, Casswell, & You, 2012). Other studies have demonstrated the same, consistent pattern: Patterson et al. (2004) reported that both higher area-level and higher individual-level social cohesion lowered the probability of smoking (Patterson, 2004); and Lindstrom (2004) observed that individual perceptions of trust (a component of social cohesion) were inversely associated with cannabis use among young adults (Lindström, 2004). Conversely, mixed and inconsistent results have been published on the influences of social cohesion on alcohol consumption (Lin et al., 2012). On one hand, Åslund and Nilsson found that low perceived social cohesion increased the odds of high alcohol consumption by 50%, and doubled the odds of smoking and illicit drug use among adolescents (Åslund & Nilsson, 2013); and Duncan, Duncan, and Strycker (2002) found that higher social cohesion decreased youth alcohol use in neighborhoods, echoing this finding (Duncan et al., 2002). On the other hand, two other studies yielded no significant correlations between individually- perceived social cohesion and alcohol intake (Lundborg, 2005; Poortinga, 2006).

In addition, social cohesion appears to be directly associated with depression (Choi, Kim, DiNitto, & Marti, 2015). Mental health inequality could potentially be reduced by strengthening social cohesion (Fone et al., 2014). In addition, there is an intertwined relationship between depression and substance use dependence. Depressed people are more likely to develop substance use dependence and people with substance use dependence problems tend to become more depressed (Boden & Fergusson, 2011; Schuckit, Smith, & Chacko, 2006; Sullivan, Fiellin, & O'Connor, 2005). Since social cohesion has been found to be associated with both substance use disorders and depression, we would like to further examine whether social cohesion has a direct effect on substance use, or an indirect effect mediated by depression.

One limitation of the aforementioned, previously-published studies is that they all were cross-sectional, which prohibits conclusions regarding causation and calls for longitudinal studies to understand the impacts of perceived social cohesion on substance use disorders. Longitudinal studies also allow for investigation into whether the relationships between social cohesion, depression and substance use severity change over time.

It is still not well examined how social cohesion and depression can influence substance use disorders (alcohol use disorder, nicotine use dependence, and cannabis use disorder) over time. Therefore, our goal was to study the effects of social cohesion on substance use severity. The two main purposes of the study were: 1) to examine for longitudinal associations between social cohesion, the severity of use of three substances, and depression; and 2) to further assess whether the hypothesized associations between social cohesion and each SUD are mediated by depression. To address these questions together, structural equation models (SEMs) were used. The current study thereby aims to extend beyond prior investigations to examine cross-sectional and longitudinal pathways between social cohesion, depression and SUDs in young Swiss men, using data from an on-going cohort study. In addition, our target group was at their peak time of life (age 21–25) for substance use.

2. Materials and methods

2.1. Recruitment procedure

For the present study, data were extracted from an ongoing cohort study — the *Cohort Study on Substance Use Risk Factors (C-SURF)* — a longitudinal study designed to assess substance use patterns and their related consequences in a cohort of young Swiss men over time. The protocol (Protocol No. 15/07) was approved by Lausanne University Medical School's Clinical Research Ethics Committee and informed written consent was obtained from all participants.

In Switzerland, army recruitment is compulsory and virtually all young men of roughly 19 years old are required to present themselves to one of six national recruitment centers, so their eligibility for the army or civil service can be determined. Study enrolment for C-SURF took place between August 23, 2010 and November 15, 2011, at three army recruitment centers, one located in a French-speaking area of Switzerland (Lausanne) and two in German-speaking areas (Windisch and Mels). These three centers encompass 21 of Switzerland's 26 cantons, including all the French-speaking cantons. As there is no pre-selection for army conscription, virtually the entire Swiss male population in this age group was eligible for inclusion in C-SURF.

2.2. Participants

Of the 7563 men who gave written consent indicating their willingness to participate (50.2% of the eligible population), 5987 completed the baseline questionnaire between September 2010 and March 2012. This questionnaire was sent out to them privately, two weeks after their recruitment-center visit. Questionnaires were self-completed, with no face-to-face contact. Sampling procedures and potential non-response bias have been described elsewhere (Studer et al., 2013). Briefly, non-respondents drank more alcohol than respondents, but the magnitude of the difference was small, indicating minimal non-response bias. Baseline data were not included in the analysis for the current study, since questions regarding social cohesion were not included in the baseline questionnaire. The mean age of participants at baseline was 20.1 years old.

The first follow-up assessment (t_1) occurred between March 2012 and January 2014, about 1.5 years after the baseline assessment (t_0). The second follow-up assessment (t_2) was conducted between April 2016 and March 2018, roughly 5.5 years after the baseline assessment. Subjects who did not undergo a baseline assessment were still invited to participate at t_1 and t_2. Individuals were included in our analysis only when they answered questionnaires at both t1 and t2, resulting in 5372 participants. Individuals who had missing answers to questions, or only participated in one wave were excluded. Missing values were deleted listwise, resulting in 4983 for AUD analyses, 4774 for NUD analyses, and 5020 for CUD analyses. We found no differences between groups with versus without missing data. A sensitivity analysis concluded with multiply-imputed data yielded equivalent substantive findings, so we present the listwise analyses for clarity and simplicity.

2.3. Variables

2.3.1. Socio-demographic variables

Socio-demographic variables — including age, language region (French- or German-speaking), and highest completed level of education — were assessed at both follow-up assessments. Highest completed level of education was categorized into three levels of schooling: primary (< 9 years); secondary (from 9 to 12 years); and beyond secondary school (13 years or more).

2.3.2. Social cohesion

At t_1, perceived social cohesion was measured using a comprehensive 16-item questionnaire designed to evaluate three categories of social cohesion: "trust", "attachment", and "tolerance" (Stafford et al., 2003). At t_2, social cohesion was measured using a shortened form of that same questionnaire, with just nine of the 16 items (Dupuis, Baggio, & Gmel, 2017). For each statement, participants were asked to use a 7-point Likert scale to indicate how strongly they agreed or disagreed: (7) very strongly agree, (6) strongly agree, (5) mildly agree, (4) neutral, (3) mildly disagree, (2) strongly disagree, and (1) very strongly disagree. In

this study, to make the two data-collection waves comparable, we selected the exact same nine items from each of the two follow-up assessments for analysis. This comprised three subscales assessing distinct cognitive aspects of neighborhood cohesion: trust (e.g., trust in people, including members of the neighborhood who are not personally known); attachment to neighborhood (e.g., feeling part of the community); and tolerance (e.g., reciprocal tolerance within the community). In this study, only the general factor (mean score) was used.

Only participants who answered more than six out of nine questions at both t_1 and t_2 were included in the analysis. We averaged scores to create a mean social cohesion score, based on the number of questions respondents answered, with average scores ranging from 1 to 7. The internal consistency of social cohesion questionnaires was excellent at t_1 (Cronbach's alpha coefficient = 0.93) and at t_2 (Cronbach's alpha coefficient = 0.92).

2.3.3. Depression

Depression was assessed at t_1 and t_2 using the World Health Organization's Major Depression Inventory (ICD-10)-WHO-MDI (Olsen, Jensen, Noerholm, Martiny, & Bech, 2003). It contains a 10-item questionnaire for which available responses are on a six-point scale, from 0 (never) to 5 (all the time). The ten items are summed up to give a total score for depression severity (range 0–50).

2.3.4. Alcohol use disorder (AUD)

Twelve questions for alcohol use dependence severity were asked about, as per Knight el al., including the additional criterion of 'craving' (Knight et al., 2002). These questions correspond to the symptoms of alcohol use dependence severity, as defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), and were assessed for the preceding 12 months, with two answer options for each criterion (yes/no). We used the summed score of the Alcohol Use Disorders Identification Test (AUDIT), which ranges from 0 to 12, to demonstrate alcohol use disorder (AUD).

2.3.5. Nicotine use dependence (NUD)

The Fagerström Test for Nicotine Dependence (FTND) was used to assess nicotine dependence (Heatherton, Kozlowski, Frecker, & Fagerström, 1991). A continuous total score ranging between 0 "very low dependence" and 10 "very high dependence" was generated.

2.3.6. Cannabis use disorder (CUD)

Cannabis use dependence severity was assessed using the Cannabis Use Disorder Identification Test (CUDIT) on a scale from 0 to 40 (Adamson & Sellman, 2003). We used the summed score for cannabis dependence severity.

2.4. Statistical analyses

All statistical analyses were conducted using STATA Special Edition 15.0 (StataCorp, Texas, USA). Descriptive statistics were calculated to characterize respondents from the two data-collection waves (data collection periods) of the C-SURF population. Social cohesion variables were tested by Wilcoxon signed rank test to examine if there was any difference between the two waves. The characteristics of baseline participants have been described elsewhere (Gmel et al., 2015). All the dependent variables were measured twice, at both follow-up 1 (t_1) and follow-up 2 (t_2)— including alcohol, nicotine, and cannabis. The continuous summed scores of these measures were used as severity scores.

2.4.1. Analytical frame

Models were constructed step by step. The first step involved creating a path model that only consists of two factors from each wave: social cohesion and SUD. We concluded that the true path was from social cohesion towards SUD, rather than from SUD towards social cohesion, based upon prior study findings (Lin et al., 2012; Patterson, 2004), and upon the significant path coefficients generated by cross-lagged panel analyses. Secondly, we ran a separate path model for depression and social cohesion, which was bi-directional. Third, we assessed for associations between depression and the three SUDs. Structural equation models were created to differentiate direct and indirect effects in a longitudinal framework. A detailed description is provided in the Supplementary document.

We conducted mediator analyses to test the direct and indirect effects via depression, when linking the two waves together. All coefficients were estimated in SEMs and, thus, can adjust all the variables at t1 and t2 simultaneously. Model fit was evaluated using the root mean square error of approximation (RMSEA $\leq$ 0.06), comparative fit index (CFI $\geq$ 0.95) and standardized root mean square residual (SRMR $\leq$ 0.08) (Hu & Bentler, 1999). Due to the non-normally distributed variables at t1 and t2, asymptotic distribution free (ADF) was chosen to produce more efficient and reliable estimates.

To screen for mediation, corrected bootstrap 95% confidence intervals (CI) were computed by means of bootstrap resampling with 5000 draws. Ninety-five percent confidence intervals around the point estimate of an indirect effect that do not cross zero indicate statistical significance.

3. Results

Descriptive statistics for the two waves of data are summarized in Table 1. The mean age of participants was 21.3 years at the time of the first follow-up and 25.4 years at the second. A little more than half were French-speaking (58%). More than half of the participants (56.3%) reported that they had a tertiary level of education at t_2. The mean score for major depression increased, whereas the mean scores for alcohol and nicotine dependence severity remained within the same range. The distributions, by percentage, of depression, AUD, NUD and CUD are shown in Supplementary Table S1. The median score for social cohesion at t1 was 5.4 (Interquartile range (IQR): 4.3–6.0) and decreased to 5.0 (IQR: 4.1–5.8). The item-by-item median social cohesion scores at each assessment point, and the 25% to 75% range for each score, are shown in Supplementary Table S2.

Fig. 1 depicts the longitudinal association between social cohesion, depression and SUDs via pathway analysis, using SEMs; this was done

Table 1
Demographic and clinical characteristics of the survey sample at each data collection point.

	1st follow-up, t_1	2nd follow-up, t_2
	N (%) or mean±SD	N (%) or mean±SD
Number of subjects	5372	5372
Age	21.3 ± 1.3	25.4 ± 1.3
Linguistic region		
French	3130 (58.3)	same
German	2242 (41.7)	same
Highest achieved education level	5356	5358
Mandatory (primary) school	477 (8.9)	241 (4.5)
Secondary school	2366 (44.2)	2100 (39.2)
Beyond secondary school	2513 (46.9)	3017 (56.3)
Major depression	5207	5284
[a] MDI score	7.9 ± 7.1	8.7 ± 7.4
Alcohol use severity	5322	5356
[b] AUDIT score	1.3 ±1.8	1.3 ±1.7
Nicotine use severity	5008	5247
[c] FTND score	0.9 ±1.7	0.9 ±1.7
Cannabis use severity	5357	5357
[d] CUDIT score	1.5±4.1	1.5 ±4.4
Social cohesion: Median (**Q1-Q3**)	**5.4 (4.3–6.0)**	**5.0 (4.1–5.8)**

[a] MDI: World Health Organization's Major Depressive Inventory (0–50).
[b] AUDIT: Alcohol Use Disorders Identification Test (0–12).
[c] FTND: Fagerstrom Test for Nicotine Dependence (0–10).
[d] CUDIT: Cannabis Use Disorders Identification Test (0–40).

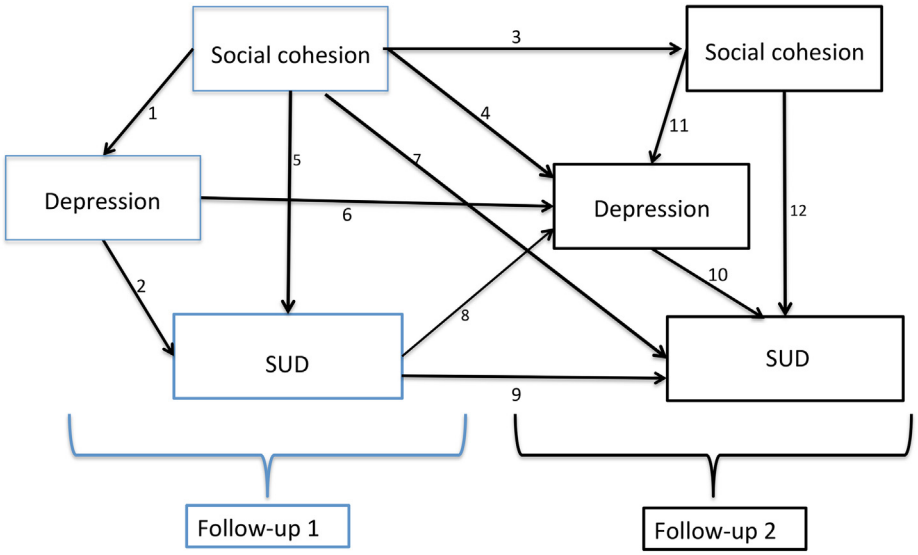

Fig. 1. Mediator pathway from social cohesion through depression on substance use disorder (SUD), including alcohol use disorder (AUD), nicotine use disorder (NUD), and cannabis use disorder (CUD), separately.

separately for AUD, NUD, and CUD.

We did not observe any significant direct effects of social cohesion on AUD, and all the associations were non-significant and standardized beta coefficients were: −0.003 (t1 → t1), 0.015 (t1 → t2), 0.019 (t2 → t2) (Table 2).

For direct effects on NUD, social cohesion at t1 was significantly associated with NUD at t1 (standardized beta = -0.082) (Table 3). Social cohesion at t1 and t2 were not associated with NUD at t2 (standardized beta = -0.007, t1 → t2 and −0.020, t2 → t2). For CUD, social cohesion was cross-sectionally associated with CUD in both waves (standardized beta = -0.085 (t1 → t1), and −0.033 (t2 → t2)), but the association from t1 to t2 was non-significant (standardized beta = 0.010 (t1 → t2)) (Table 4). The standardized beta coefficients for the association between social cohesion and each SUD (AUD, NUD, CUD) between t1 and t2 are 0.480, 0.679, and 0.617, respectively.

For mediation effects, the direct effects from social cohesion on AUD were all not significant, whereas the indirect effects through depression were significant and negative. The social cohesion has direct and significant effects on NUD at t1, but not at t2 or longitudinal direct effects (from t1 to t2). Social cohesion has indirect and negative effects on NUD at t1, t2, and from t1 to t2. For CUD, the direct effects of social cohesion on CUD cross-sectionally (at t1 and t2 separately) were

Table 2
Total, direct, and indirect effects from Structural Equation Models (SEMs) for alcohol use disorder (AUD).

Total effect	Path No.	beta	B	SE	p-value	95% CI	
SC at t1 → SC at t2	3	**0.382**	**0.356**	**0.013**	**< 0.001**	**0.331, 0.381**	RMSEA = 0.035
SC at t2 → AUD at t2	12	−0.005	−0.008	0.023	0.723	−0.052, 0.036	CFI = 0.985
Depression at t2 → AUD at t2	10	**0.161**	**0.037**	**0.004**	**< 0.001**	**0.030, 0.045**	SRMR = 0.016
AUD at t1 → AUD at t2	9	**0.480**	**0.471**	**0.019**	**< 0.001**	**0.434, 0.508**	
SC at t1 → AUD at t2	7	**−0.038**	**−0.057**	**0.023**	**0.011**	**−0.102, −0.013**	
SC at t2 → Depression at t2	11	**−0.150**	**−1.035**	**0.109**	**< 0.001**	**−1.248, −0.822**	
AUD at t1 → Depression at t2	8	0.027	0.116	0.060	0.056	−0.003, 0.234	
Depression at t1 → Depression at t2	6	**0.406**	**0.425**	**0.020**	**< 0.001**	**0.387, 0.464**	
SC at t1 → Depression at t2	4	**−0.220**	**−1.418**	**0.105**	**< 0.001**	**−1.624, −1.212**	
Depression at t1 → AUD at t1	2	**0.199**	**0.049**	**0.005**	**< 0.001**	**0.039, 0.059**	
SC at t1 → AUD at t1	5	**−0.053**	**−0.080**	**0.023**	**0.001**	**−0.126, −0.035**	
SC at t1 → Depression at t1	1	**−0.249**	**−1.528**	**0.107**	**< 0.001**	**−1.738, −1.319**	

Path No.	Direct effect					Indirect effect				
	beta	B	SE	p-value	95% CI	beta	B	SE	p-value	95% CI
3	**0.382**	**0.356**	**0.013**	**< 0.001**	**0.331, 0.381**	–	–	–	–	–
12	0.019	0.031	0.022	0.171	−0.013, 0.075	**−0.024**	**−0.039**	**0.005**	**< 0.001**	**−0.049, −0.028**
10	**0.161**	**0.037**	**0.004**	**< 0.001**	**0.030, 0.045**	–	–	–	–	–
9	**0.475**	**0.467**	**0.019**	**< 0.001**	**0.430, 0.504**	0.004	0.004	0.002	0.064	0.000, 0.009
7	0.015	0.022	0.020	0.263	−0.017, 0.062	−0.053	**−0.080**	**0.015**	**< 0.001**	**−0.110, −0.050**
11	**−0.150**	**−1.035**	**0.109**	**< 0.001**	**−1.248, −0.822**	–	–	–	–	–
8	0.027	0.116	0.060	0.056	−0.003, 0.234	–	–	–	–	–
6	**0.400**	**0.420**	**0.020**	**< 0.001**	**0.380, 0.459**	0.005	0.006	0.003	0.057	0.000, 0.012
4	**−0.062**	**−0.398**	**0.101**	**< 0.001**	**−0.596, −0.200**	−0.158	**−1.020**	**0.068**	**< 0.001**	**−1.152, −0.887**
2	**0.199**	**0.049**	**0.005**	**< 0.001**	**0.039, 0.059**	–	–	–	–	–
5	−0.003	−0.005	0.022	0.822	−0.049, 0.039	−0.050	**−0.075**	**0.009**	**< 0.001**	**−0.093, −0.058**
1	**−0.249**	**−1.528**	**0.107**	**< 0.001**	**−1.738, −1.319**	–	–	–	–	–

Note. RMSEA, root mean square error of approximation; CFI, comparative fit index; SRMR, standardized root mean square residual; SC, social cohesion; beta, standardized coefficient; B, unstandardized coefficient; SE, standard error of B; CI, confidence interval of B.

Table 3
Total, direct, and indirect effects from Structural Equation Models (SEMs) for nicotine use disorder (NUD).

Total effect	Path No.	beta	B	SE	p-value	95% CI	
SC at t1 → SC at t2	3	**0.378**	**0.351**	**0.015**	**< 0.001**	**0.322, 0.381**	RMSEA = 0.035 CFI = 0.986 SRMR = 0.016
SC at t2 → NUD at t2	12	**−0.029**	**−0.046**	**0.020**	**0.023**	**−0.085, −0.006**	
Depression at t2 → NUD at t2	10	**0.064**	**0.015**	**0.003**	**< 0.001**	**0.008, 0.021**	
NUD at t1 → NUD at t2	9	**0.679**	**0.684**	**0.017**	**< 0.001**	**0.652, 0.717**	
SC at t1 → NUD at t2	7	**−0.103**	**−0.149**	**0.022**	**< 0.001**	**−0.193, −0.106**	
SC at t2 → Depression at t2	11	**−0.148**	**−1.024**	**0.109**	**< 0.001**	**−1.238, −0.810**	
NUD at t1 → Depression at t2	8	0.066	**0.292**	**0.065**	**< 0.001**	**0.165, 0.419**	
Depression at t1 → Depression at t2	6	**0.413**	**0.433**	**0.020**	**< 0.001**	**0.394, 0.473**	
SC at t1 → Depression at t2	4	**−0.224**	**−1.436**	**0.107**	**< 0.001**	**−1.645, −1.227**	
Depression at t1 → NUD at t1	2	**0.108**	**0.026**	**0.004**	**< 0.001**	**0.017, 0.034**	
SC at t1 → NUD at t1	5	**−0.109**	**−0.158**	**0.022**	**< 0.001**	**−0.201, −0.115**	
SC at t1 → Depression at t1	1	**−0.253**	**−1.548**	**0.108**	**< 0.001**	**−1.761, −1.336**	

Path No.	Direct effect					Indirect effect				
	beta	**B**	**SE**	**p-value**	**95% CI**	**beta**	**B**	**SE**	**p-value**	**95% CI**
3	**0.378**	**0.351**	**0.015**	**< 0.001**	**0.322, 0.381**	–	–	–	–	–
12	−0.020	−0.031	0.020	0.123	−0.070, 0.008	**−0.010**	**−0.015**	**0.004**	**< 0.001**	**−0.022, −0.008**
10	**0.064**	**0.015**	**0.003**	**< 0.001**	**0.008, 0.021**	–	–	–	–	–
9	**0.675**	**0.680**	**0.017**	**< 0.001**	**0.647, 0.713**	**0.004**	**0.004**	**0.001**	**< 0.001**	**0.002, 0.007**
7	−0.007	−0.010	0.019	0.590	−0.046, 0.026	**−0.096**	**−0.139**	**0.018**	**< 0.001**	**−0.174, −0.105**
11	**−0.148**	**−1.024**	**0.109**	**< 0.001**	**−1.238, −0.810**	–	–	–	–	–
8	**0.066**	**0.292**	**0.065**	**< 0.001**	**0.165, 0.419**	–	–	–	–	–
6	**0.406**	**0.426**	**0.020**	**< 0.001**	**0.386, 0.466**	**0.007**	**0.007**	**0.002**	**< 0.001**	**0.003, 0.012**
4	**−0.058**	**−0.370**	**0.103**	**< 0.001**	**−0.573, −0.168**	**−0.166**	**−1.066**	**0.070**	**< 0.001**	**−1.202, −0.929**
2	**0.108**	**0.026**	**0.004**	**< 0.001**	**0.017, 0.034**	–	–	–	–	–
5	**−0.082**	**−0.119**	**0.022**	**< 0.001**	**−0.162, −0.075**	**−0.027**	**−0.040**	**0.007**	**< 0.001**	**−0.053, −0.026**
1	**−0.253**	**−1.548**	**0.108**	**< 0.001**	**−1.761, −1.336**	–	–	–	–	–

Note. RMSEA, root mean square error of approximation; CFI, comparative fit index; SRMR, standardized root mean square residual; SC, social cohesion; beta, standardized coefficient; B, unstandardized coefficient; SE, standard error of B; CI, confidence interval of B.

Table 4
Total, direct 3and indirect effects from Structural Equation Models (SEMs) for cannabis use disorder (CUD).

Total effect	Path No.	beta	B	SE	p-value	95% CI	
SC at t1 → SC at t2	3	**0.384**	**0.351**	**0.015**	**< 0.001**	**0.322, 0.381**	RMSEA = 0.040 CFI = 0.978 SRMR = 0.018
SC at t2 → CUD at t2	12	**−0.047**	**−0.187**	**0.051**	**< 0.001**	**−0.286, −0.087**	
Depression at t2 → CUD at t2	10	**0.092**	**0.053**	**0.010**	**< 0.001**	**0.033, 0.073**	
CUD at t1 → CUD at t2	9	**0.617**	**0.666**	**0.035**	**< 0.001**	**0.597, 0.735**	
SC at t1 → CUD at t2	7	**−0.095**	**−0.355**	**0.056**	**< 0.001**	**−0.465, −0.245**	
SC at t2 → Depression at t2	11	**−0.151**	**−1.040**	**0.108**	**< 0.001**	**−1.252, −0.827**	
CUD at t1 → Depression at t2	8	**0.057**	**0.106**	**0.031**	**0.001**	**0.046, 0.167**	
Depression at t1 → Depression at t2	6	**0.405**	**0.425**	**0.020**	**< 0.001**	**0.386, 0.465**	
SC at t1 → Depression at t2	4	**−0.222**	**−1.429**	**0.104**	**< 0.001**	**−1.633, −1.226**	
Depression at t1 → CUD at t1	2	**0.128**	**0.072**	**0.011**	**< 0.001**	**0.050, 0.094**	
SC at t1 → CUD at t1	5	**−0.117**	**−0.404**	**0.055**	**< 0.001**	**−0.512, −0.296**	
SC at t1 → Depression at t1	1	**−0.250**	**−1.532**	**0.107**	**< 0.001**	**−1.742, −1.322**	

Path No.	Direct effect					Indirect effect				
	beta	**B**	**SE**	**p-value**	**95% CI**	**beta**	**B**	**SE**	**p-value**	**95% CI**
3	**0.384**	**0.351**	**0.015**	**< 0.001**	**0.322, 0.381**	–	–	–	–	–
12	**−0.033**	**−0.132**	**0.051**	**0.010**	**−0.232, −0.031**	**−0.014**	**−0.055**	**0.012**	**< 0.001**	**−0.079, −0.032**
10	**0.092**	**0.053**	**0.010**	**< 0.001**	**0.033, 0.073**	–	–	–	–	–
9	**0.612**	**0.660**	**0.035**	**< 0.001**	**0.592, 0.729**	**0.005**	**0.006**	**0.002**	**0.003**	**0.002, 0.009**
7	0.010	0.036	0.048	0.456	−0.058, 0.129	**−0.105**	**−0.390**	**0.047**	**< 0.001**	**−0.483, −0.298**
11	**−0.151**	**−1.040**	**0.108**	**< 0.001**	**−1.252, −0.827**	–	–	–	–	–
8	**0.057**	**0.106**	**0.031**	**0.001**	**0.046, 0.167**	–	–	–	–	–
6	**0.398**	**0.418**	**0.020**	**< 0.001**	**0.378, 0.457**	**0.007**	**0.008**	**0.002**	**0.002**	**0.003, 0.012**
4	**−0.058**	**−0.373**	**0.101**	**< 0.001**	**−0.572, −0.175**	**−0.164**	**−1.056**	**0.068**	**< 0.001**	**−1.189, −0.924**
2	**0.128**	**0.072**	**0.011**	**< 0.001**	**0.050, 0.094**	–	–	–	–	–
5	**−0.085**	**−0.294**	**0.056**	**< 0.001**	**−0.403, −0.185**	**−0.032**	**−0.110**	**0.018**	**< 0.001**	**−0.146, −0.075**
1	**−0.250**	**−1.532**	**0.107**	**< 0.001**	**−1.742, −1.322**	–	–	–	–	–

Note. RMSEA, root mean square error of approximation; CFI, comparative fit index; SRMR, standardized root mean square residual; SC, social cohesion; beta, standardized coefficient; B, unstandardized coefficient; SE, standard error of B; CI, confidence interval of B.

significant and negative, but not longitudinally (from t1 to t2). Similar to AUD and NUD, the indirect effects were significant and negative (Table 4).

4. Discussion

This study demonstrates cross-sectional and longitudinal associations between social cohesion and problematic substance use in a large general population sample of young Swiss men. Social cohesion, depression and substance use dependence were heavily intertwined throughout the two waves of our survey. The longitudinal effects of social cohesion on substance use severity were partially mediated by depression. Once a person develops problematic substance use at an early stage of life, depression plays a more important role in his problematic substance use than social cohesion does. Our study extends the existing literature by identifying neighborhood social cohesion as an additional predictor of problematic substance use in early adulthood.

In our study, social cohesion did not directly affect alcohol use disorders. Our findings echo findings reported by Ruiz et al (Ruiz et al., 2019). Social cohesion has been found to be associated not with AUDs/DUDs (drug use disorders) (Savage & Mezuk, 2014), but with hazardous drinking (Lin et al., 2012). In our study, social cohesion did not have direct effects on problematic alcohol use, but exhibited indirect effects through depression. Consuming large amounts of alcohol is more common in early adulthood and drinking is more encouraged in some societies (Fujimoto & Valente, 2012; Kuntsche & Gmel, 2013). This indicates that personal depression plays a more important role in problematic alcohol use. Social cohesion acted as one mechanism for alcohol drinking, and as another mechanism for smoking.

One hypothesis explaining how social cohesion influences health behaviors includes four components: the sharing of information, the establishment of norms, the exertion of social controls, and the strengthening of psychosocial resources (reducing stress) (Lin et al., 2012; Patterson, 2004). Patterson et al. have found that increased social cohesion results in the increased sharing of health-related information pertaining to, among other things, smoking consequences. Discouraging the use of tobacco could help to establish a social norm. In addition, social cohesion also provides psychosocial support which may reduce distress, a known risk factor for cigarette smoking and cannabis use (Patterson, 2004). The negative association between social cohesion and nicotine use and problematic cannabis use could be explained as individuals who are from more cohesive community feeling more integrated within and more watched by others and, hence, being more likely to refrain from health-damaging behaviors (Lin et al., 2012).

Depression is associated with concurrent alcohol use and impairment and with drug use and impairment (Conner, Pinquart, & Gamble, 2009). Depression has also been found to be associated with substance use disorders in several large epidemiological studies (Conway, Compton, Stinson, & Grant, 2006; Grant et al., 2015; Lai, Cleary, Sitharthan, & Hunt, 2015). When investigating the relationship between social cohesion and problematic substance use, we have found that depression is a partial mediator. One potential explanation for how social cohesion might influence substance use is that greater social cohesion is associated with less depression, which further lowers the likelihood of problematic substance use. Higher-level social cohesion was associated with lesser levels of depression in one US aging study (Choi et al., 2015). Similarly, in a study within the United Kingdom (UK), higher-level social cohesion was shown to reduce adverse mental effects (Fone et al., 2014). The significant indirect pathways via depression and problematic substance use at an earlier age in our study echo these findings.

It is difficult to compare our results directly with other studies because different measures of social cohesion and depression were used. Two European studies have shown that social cohesion was associated with later depression among adults (Ruiz, Scholes, & Bobak, 2018; Urzua et al., 2019). The first study showed that participants with low social cohesion were 44% significantly more likely to have incident probable depression over 12 years, than those counterparts of high social cohesion. However, this study recruited only participants who were older than 50 years old (Ruiz et al., 2018). The second study showed participants with lower social cohesion (lowest tertile vs highest tertile) were more likely to have elevated depressive symptoms (Odds ratios = 1.33) (Urzua et al., 2019).

The effect size of those two studies were found to be small (Cohen's $d < 0.2$ when $OR < 1.5$) (Chen, Cohen, & Chen, 2010). Our study showed a similar trend, i.e. increased social cohesion was associated with decreased depressive score. One standard deviation (SD) increase of social cohesion score is associated with −0.15 SD decrease of depressive score (standardized coefficient of −0.15). Based on Peterson and Brown's suggested procedure, the standardized beta coefficients (-0.15) can be directly converted to Pearson correlation (r = -0.2) (Lenhard & Lenhard, 2017; Peterson & Brown, 2005). The effect size of 0.2 might be considered as small to medium range, according to Cohen's criteria (1988) (Cohen, 1988). However, when looking at the relationship between social cohesion and substance use disorder, depression plays an important role as mediator. Therefore, even a small effect size may be meaningful, from a public health perspective (Durlak, 2009).

In the current study, depression was a mediator in the association between social cohesion and substance use disorders. Depressed individuals have been more likely to develop problematic alcohol use in some studies (Cooper, Frone, Russell, & Mudar, 1995; Schick, Weiss, Contractor, Dixon-Gordon, & Spillane, 2019; Schuckit et al., 2006). However, other authors have also proposed that chronic drinking could promote depression indirectly, potentially due to a stressful life or difficult partner relationships (Boden & Fergusson, 2011; Sullivan et al., 2005). The later pathway direction was not identified in our study, which could be due to a less stressful life or the lack of established partner relationships over the course of our study period.

Only a few published studies have investigated social cohesion and its impact on substance use disorders. In one cross-sectional US study, neighborhood cohesion was not related to AUD or to DUD; but neighborhood safety and family cohesion were protective against AUD/DUD among Latino and Asian-American immigrants (Savage & Mezuk, 2014). The average age in that study was 36–39 years old, whereas our study participants were much younger (21–25 years old). The prevalence of AUD/DUDs in that study also was relatively low (4.1% for Asian Americans and 2.7% for adult immigrants), which might have limited the study's statistical power to detect true associations. In our cohort, roughly 33%, 11% and 8% met the criteria for AUD, NUD, and CUD, respectively. Additionally, neighborhood characteristics were indexed by seven items in their study and separated into two factors — ¨social cohesion¨ and ¨social safety¨ — whereas social cohesion included nine items in our study. One group of investigators concluded that SUDs mediate the association between social capital (i.e., participation in social activities and perceptions of relationships) and depression symptoms (Awgu, Magura, & Coryn, 2016). To expand on their conclusion, a longitudinal design is needed to determine long-term connections between different factors. Our data provided the potential to investigate pathway directions and supported that depression is the mediator. Importantly, social capital and social cohesion have different definitions in social science (Cloete, 2014).

We acknowledge that, among males, moderate social cohesion is associated with more hazardous alcohol use (OR = 1.29, 1.08–1.53) (Kuipers, van Poppel, van den Brink, Wingen, & Kunst, 2012). Lin et al. found that residents who perceived their neighborhood as more cohesive reported a higher annual frequency of alcohol consumption, but less consumption during a typical drinking occasion (Lin et al., 2012). In our study, social cohesion did not exert direct effects on problematic alcohol use, but the person's own depression level did influence it.

All the aforementioned studies also were cross-sectional, rather than longitudinal in design. Using longitudinal data allowed us to control for

substance use severity and other pathways at the first assessment. Longitudinal studies have been published examining the association between social cohesion and other outcomes — like mental health and the frequency of going out among older adults (Choi et al., 2015; Fone et al., 2014).

To our knowledge, no longitudinal study has previously analyzed the relationship between social cohesion, depression and substance use severity among adults. In young adults, the protective effect of perceiving more social cohesion might lower levels of stress and anxiety, and increase trust in social relationships, reducing their stress and thereby removing one major motivation that many have to use substances heavily (Lin et al., 2012).

We are aware that the within wave mediation might lead to a degree of bias (Maxwell & Cole, 2007); however, our results suggest that depression plays an important mediating role over the long term. Thus, we assume that this mediator role could also be seen within a wave. The temporal order of depression and substance use disorder was also based on prior research and theory (Ruiz et al., 2018; Urzua et al., 2019). This fundamental limitation might be overcome in future studies, when there are repeated measurements at three waves.

The current study has limitations. First, our sample is representative of Swiss young men only, and further studies must be conducted to investigate whether our findings can be reproduced in females and older adults. Second, we assessed individual perceptions of neighborhood cohesion, which might not reflect the actual levels of community cohesion that exist. We also acknowledge that not all social-related predictors, such as social capital or family cohesion, were captured in our study.

5. Conclusions

In conclusion, among men in their early 20s, higher-level social cohesion appears to have a protective effect against severe substance use. The longitudinal effects of social cohesion on substance use appear to be partially mediated by depression. These findings have the potential to guide the development of strategies for substance use dependence prevention. Social cohesion plays more important role at an early age, while depression has a long-term role. Social cohesion among neighbors may buffer against individual and other risk factors.

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7. Contributors

D-H. Tsai proposed the analysis, analysed the data, and wrote the initial draft and final version of the article. S. Foster participated in data analyses, reviewed the article and provided feedback throughout the writing period. G. Gmel designed the study, reviewed the article and provided feedback throughout the writing period. M. Mohler-Kuo designed the study, discussed earlier versions, edited the text, and reviewed the final version of the article. All authors contributed to and have approved the final manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.addbeh.2020.106510.

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